

平成 31 年 1 月 29 日  
January 29, 2019

大学院学生各位  
To All Graduate Students

平成 30 年度  
基盤医学特論 開講通知  
Information on Special Lecture Tokuron 2018.4-2019.3

**Title : EPIGENETICS, LIFE BEYOND YOUR GENES: IMPLICATIONS IN  
BEHAVIOR AND HEALTH**

Teaching Staff : Tapas K. Kundu, Ph.D.  
(Professor, Transcription and Disease Laboratory, Molecular Biology and Genetics Unit,  
Jawaharlal Nehru Centre for Advanced Scientific Research)

日時 : 平成 31 年 3 月 8 日(金) 17 時 00 分より (90 分)  
Time and Date : March 8 (Fri.), 2019 17 : 00~ (90 minutes)

場所 : 基礎研究棟 1 階 会議室 1  
Room : Meeting Room 1 (Basic Medical Research Building, 1st floor)

言語 : 英語  
Language : English

The DNA sequence of living organisms codes for RNA and is referred to as 'Gene'. However, our gene sequence alone cannot determine the regulation of gene expression (function) during development, signal response and disease manifestation. Small chemical modifications of gene (DNA sequence) and its associated proteins, such as DNA methylation, histone methylation, acetylation, phosphorylation etc. regulate the gene function on and above the gene sequence. Therefore, it is termed as 'Epigenetics'. Epigenetics is related to not only our tissue typing but also all the activities, behavior and ability to fight diseases. We do inherit some epigenetic modifications, and this phenomenon is referred to as 'Transgenerational Epigenetics'. But most of the epigenetic modification are trans-cellular and are closely linked to different diseases which are not hereditary, including cancer, a few neurological disorders, drug addiction or alcoholism. Significantly, even in case of infectious diseases, epigenetic modifications play crucial role in the virulence and disease establishment. Therefore, the enzymes which are involved in epigenetic modifications have become important targets of therapeutic molecules today. We have discovered several small molecule modulators of epigenetic enzymes and shown their possible therapeutic potential. Recently, our laboratory has discovered a natural compound derived small molecule TTK21, specific activator of the acetyltransferase KAT3 family (p300/CBP). We have shown that this activator can induce neurogenesis in the mouse brain and thereby enhance the formation of long term memory. We have demonstrated that synaptic plasticity and memory deficiencies can be restored in a mouse model of tauopathy following treatment with TTK21 conjugated to glucose derived carbon nanosphere (CSP). By employing the CSP-TTK21, we could selectively reverse epigenetic, transcriptional, synaptic plasticity, and behavioral deficits associated with an Alzheimer's disease-related disorder. The effectiveness of the CSP-TTK21 is currently being studied in the other neurological disorders and depression.

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Contact : Nami Takagi, Division of Cancer Biology (Ext. 2463)

事前の申込みは不要です。No Registration required.  
講義開始後 30 分迄に御入室下さい。Please take a seat before 17:30  
途中退室不可 Please stay until the end of the lecture.